infiltrate (neutrophils and lymphocytes), air spaces deformed by organizing connective tissue plugs, and foamy macrophages.

Ticlopidine therapy was withdrawn; prednisone was given in the same schedule and dose. The patient subsequently felt better, and dyspnea and blanching cutaneous lesions improved. Dyspnea disappeared in 3 months, and the interstitial pattern completely resolved in 5 months.

Bronchiolitis obliterans–organizing pneumonia is a clinical pathologic syndrome with a wide spectrum of causes, including idiopathic and secondary causes (4). The latter are associated with several connective tissue diseases, inhaled substances and fumes, radiation therapy, and hematologic diseases (4). No ticlopidine-related pulmonary effects have been reported. Increased CD8 lymphocyte counts and the communicated augmented respiratory-burst metabolism in human neutrophils caused by ticlopidine (5) seem to have contributed to the pathogenesis of lung injury in our patient.

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Reversible Dysgeusia Attributed to Losartan

To the Editor: Like angiotensin-converting enzyme (ACE) inhibitors, losartan interferes with the renin–angiotensin–aldosterone system by decreasing angiotensin II–mediated effects. Although losartan and ACE inhibitors have similar therapeutic potency, losartan reportedly has fewer adverse effects because of selective antagonism of angiotensin I receptors (1). Schlienger and colleagues (2) recently described a patient in whom losartan induced reversible ageusia; we present two similar reports.

A 49-year-old woman had been using enalapril (10 mg/d) for the treatment of hypertension. Because of fatigue, therapy was changed to losartan (50 mg/d). One week after the initiation of therapy, the patient reported a persistent metallic taste, a tickling cough, and intestinal symptoms. After discontinuation of losartan therapy, symptoms disappeared. Concomitant medications were carbaspirin calcium (38 mg/d), cetirizine (10 mg/d), and ranitidine (150 mg three times daily).

A 69-year-old woman had been using perindopril (4 mg/d) for the treatment of hypertension. Because of a tickling cough, therapy was changed to losartan (10 mg/d). After 3 months, the patient developed a burning feeling on the tongue and a complete loss of taste. Perindopril therapy was restarted, and the taste disturbances disappeared within 1 week. Concomitant medications were bemetanide (1 mg three times daily) and acenocoumarole (1 mg) as prescribed.

The temporal association and the lack of suspected concomitant medication suggests a causal relation between dysgeusia and the use of losartan. We contacted the manufacturer and found that 11 cases of dysgeusia and 1 case of ageusia had been reported through a safety monitoring program. Dysgeusia is also associated with valsartan, another angiotensin II antagonist (3). The mechanism underlying losartan-induced dysgeusia is unknown. Taste disturbances induced by ACE inhibitors have tentatively been ascribed to chelation of metal ions, such as zinc (4). Losartan, however, is not known to have chelating properties. Our observation of dysgeusia during the use of losartan but not during the use of ACE inhibitors in the same patient suggests a different pharmacologic mechanism for the two phenomena.

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Acute Intravascular Hemolysis after Pollen Ingestion

To the Editor: Pollen sensitization occurs through contact with respiratory allergens. Anaphylactic shock or urticaria induced by pollen ingestion has been described in cases of atopy (1). Hemolytic anemia has occurred after ingestion of plants or drugs containing flavonoids (2–4). We report what we believe to be the first case of immunoallergic hemolytic anemia after repeated self-administration of pollen.

A 45-year-old woman had venous insufficiency treated in 1994 by troxerutine, 3500 mg. She had eaten royal jelly for 5 years and had eaten pollen grains for 2 weeks of the year for 2 years. In October 1996, transient fever, arthralgia, and lumbar pain developed after she ingested 15 g of pollen. A similar episode occurred 1 week later after a second ingestion. Two hours after a third ingestion, sudden pain developed in the back and abdomen, with fever, fatigue, jaundice, and dark urine. Anemia (iron level, 6.7 g/dL) with macrocytosis (120 fL) but no schizocytes was detected; leukocyte count was 38.5 g/L, thrombocyte count was 118 g/L, prothrombin time was 18.7 seconds (for a free hemoglobin level of 12.4 μmol/L), fibrinogen level was 1.2 g/L, level of products of fibrinogen degradation was 640 μg/mL, free hemoglobin level was 220 μmol/L, total bilirubin level was 95 mg/L (free bilirubin level, 94 mg/L), and haptoglobin level was 0.08 g/L. Electrophoresis for hemoglobinemia had normal results. No deficiency of G6PD or pyruvate kinase was found. Result of the Coombs test was positive for IgG and complement, and circulating immune complexes were found. Aspartate aminotransferase level was 340 IU/L, alanine aminotransferase level was 101 IU/L, blood urea nitrogen level was 19.98 mmol/L, and serum creatinine level was 291.7 μmol/L.

Methylprednisolone and 8 units of packed red blood cells were given, and plasma exchange was done with five sessions of hemodialysis. Hemostasis improved in 2 days, results of hepatic function tests returned to normal in 4 days, and hemodialysis was stopped 10 days later. The patient was discharged with moderate renal insufficiency 21 days after symptom onset. At 2-month follow-up, recovery was total.

Analysis of the pollen showed a heterogenous mixture of pure pollen. Results of skin-prick tests (done with total pollen, purified pollen, and routine airborne allergens) were negative. Tests for IgE and specific IgE reaction to graminae, tree, and herbacea pollen had negative results. The recurrent episodes and the positive result on the Coombs test suggested an immunoallergic reaction. The patient did not have atopy or autoimmune systemic disease, and she was not receiving any drugs. This sensitization was IgG-mediated, incriminating the ingested pollen.